

**Amendments in the specification**

*Please replace the paragraph beginning at page 1, line 1, as follows:*

This is a continuation of U.S. application Serial No. 09/635,249, filed ~~August 9,~~  
August 7, 2000, now abandoned, which is a continuation application of U.S. application Serial  
No. 08/486,546, filed May 24, 1995, now abandoned, which is a ~~continuation-in-part~~ divisional  
application of U.S. application Serial No. 08/172,329 filed December 21, 1993, now U.S. Patent  
No. 6,218,148, which is a continuation of U.S. application Serial No. 07/982,255 filed November  
25, 1992, now U.S. Patent No. 6,204,363, which is a continuation of U.S. application Serial No.  
07/684,535 filed April 10, 1991, now abandoned, which is a continuation-in-part of U.S.  
application Serial No. 07/589,701 filed October 1, 1990, now abandoned, which is a  
continuation-in-part application of U.S. application Serial No. 07/573,616 filed August 24, 1990,  
now abandoned, which is a continuation-in-part application of U.S. application Serial No.  
07/537,198 filed June 11, 1990, now abandoned, which is a continuation-in-part application of  
U.S. application Serial No. 07/422,383 filed October 16, 1989, now abandoned, each of which  
are hereby incorporated by reference.

*Please replace the paragraph beginning at page 11, line 24, as follows:*

~~Figure 24 shows~~ Figures 24A-24B show the effect of recombinant rat SCF on curing  
the macrocytic anemia of Steel mice.

*Please replace the paragraph beginning at page 12, line 4, as follows:*

~~Figure 29 shows~~ Figures 29A-29B show the effect of recombinant human sequence  
SCF treatment of normal primates in increasing peripheral WBC count.

*Please replace the paragraph beginning at page 12, line 8, as follows:*

~~Figure 30 shows~~ Figures 30A-30B show the effect of recombinant human sequence SCF treatment of normal primates in increasing hematocrits and platelet numbers.

*Please replace the paragraph beginning at page 13, line 26, as follows:*

~~Figure 42 shows~~ Figures 42A-42D show human SCF cDNA sequence (SEQ ID NOs: 60 and 61) obtained from the HT1080 fibrosarcoma cell line.

*Please replace the paragraph beginning at page 13, line 33, as follows:*

~~Figure 44 shows~~ Figures 44A-44C show human SCF cDNA sequence (SEQ ID NOs: 62 and 63) obtained from the 5637 bladder carcinoma cell line.

*Please replace the paragraph beginning at page 15, line 10, as follows:*

~~Figure 56 shows~~ Figures 56A-56B show 5-FU effect on ACH+ cells in marrow.

*Please replace the paragraph beginning at page 24, line 21, as follows:*

Isoforms of SCF are isolated using standard techniques such as the techniques set forth in commonly owned U.S. Ser. No. 421,444, now abandoned, entitled Erythropoietin Isoforms, filed October 13, 1989, hereby incorporated by reference.

*Please replace the paragraph beginning at page 182, line 6, as follows:*

Plasmid constructions for expression of numerous SCF analogs and fragments have been made. Site-directed mutagenesis had been used to prepare plasmids with initiating methionine codon followed by codons for amino acids 1 to 178, 173, 168, 166, 163, 162, 161, 160, 159, 158, 157, 156, 148, 145, 141, 137, using the numbering of FIG. 15C. The DNA for human SCF<sup>1-183</sup> (Example 6B) was cloned into MP11 from Xba1 to BamH1. Phage from this cloning was used to transfect an E. coli dut<sup>-</sup> ung<sup>-</sup> strain, R21032. Single stranded M13 DNA was prepared from this strain and site-directed mutagenesis was performed (reference IL-2 patent). After the site-directed mutagenesis reactions, the DNAs were transformed into an E. coli dut<sup>+</sup> ung<sup>+</sup> strain, JM101. Clones were screened and sequences as described in copending

U.S. application Ser. No. 717,334, filed March 29, 1985, now abandoned. Plasmid DNA preps were made from positive clones and the SCF regions from Xba1 to BamH1 were cloned into pCFM1656 as described in copending U.S. patent application Ser. No. 501,904, filed March 29, 1990, now abandoned. The oligonucleotides for each cloning were designed to substitute a stop codon for an amino acid codon at the appropriate position for each analog.